# Atopic Dermatitis—Etiology and Clinical Management

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*SUMMARY*

*Atopic dermatitis is primarily an allergic problem, similar in most of its aspects to that of allergic bronchial asthma. It should be studied and treated like asthma, with especial emphasis on the significance of inhalant allergens. It is doubtful that psychic factors are of importance in primary genesis of the disease although they may be involved in exacerbation of it.*

FEW allergic conditions seem to be so difficult of management as is atopic dermatitis, also known as allergic eczema. Formerly considered a strictly dermatologic problem, it was included in the dermatologic scrapheap of eczemas and treated as such. In the absence of other explanations as to the cause of the disease, psychogenic factors often were considered paramount, as indicated by the designation of neurodermatitis. Beginning in 1932, however, through the efforts of Sulzberger, Hill, and others, the relationship of this condition to allergic reaction was clearly established. The evidence was the frequent association or alternation of atopic dermatitis with asthma or hay fever, the high incidence of family history of allergic sensitivity, the frequency of associated eosinophilia, the frequent occurrence of positive reaction to skin tests with food and inhalant allergens, and the clinical demonstration of the specific etiologic relationship of some allergens, notably foods, to the dermatitis.

Upon the basis of this evidence, Sulzberger and Coca[8] designated this condition as *atopic dermatitis* to indicate an inflammatory condition of the skin appearing in an individual who was, by virtue of heredity, predisposed to specific sensitization. According to these criteria, then, atopic dermatitis may be described as an acute, subacute or more often inflammatory disease of the skin involving especially the flexures and characterized by intense itching, papulation, thickening and lichenification of the involved areas. It is associated with specific sensitizations to "protein allergens" responsible for the development of characteristic forms of clinical disease in an atopic patient.

Following these reports, numerous and definite demonstrations of the allergic character of this condition were offered. The etiologic relationship of allergic reaction to foods, especially in infants and young children, was definitely established by Hill[4] and others. But as the patients were studied further, it soon became evident that this was not true for older children and adults; also that elimination of foods giving specific positive skin tests and even demonstrable circulating antibodies or reagins often failed to relieve the condition. This naturally has led to considerable skepticism as to the value of skin tests in atopic dermatitis. It also has invoked the consideration of other possible etiologic factors, notably psychogenic or infectious causes, recalling the era before 1932 when the disease was considered strictly a dermatologic problem to be treated by local applications, by non-specific therapy or by psychogenic means. No wonder many practitioners have been discouraged about treating patients with the disease. This discouragement is shared not only by general practitioners, pediatricians and dermatologists, but even by many prominent allergists. Thus for example, Cooke[2] in a recent monograph stated, "Evidence is also present for my belief that allergens producing only wheal reactions do not cause dermatitis and therefore skin testing for immediate wheal reactions is not a correct procedure for an etiologic diagnosis of these exudative skin lesions." It is not surprising, therefore, that many allergists and dermatologists are so confused as to the cause of this disease, the value of skin tests and especially as to the clinical management of patients. Fortunately, this has been contrary to the author's experience.

One of the chief reasons for this confusion appears to be the assumption that food allergens are the most important causative agents. Hence, when the elimination of foods to which the patient had positive reaction did not relieve the condition, it was assumed that skin tests were valueless. For some unknown reason, little or no attention has been given to the importance of inhalant allergens, even though many allergists, including Sulzberger, Feinberg, Figley, Rowe and others, have shown an etiological relationship between such allergens as house dust, silk, flour, horse dander and pollen and atopic dermatitis in susceptible patients. In order to further emphasize the significance of this relationship, the author recently reported[10] the results of a study of 54 patients with atopic dermatitis. Upon the basis of clinical and skin test evidence, this study showed that house dust, pollens (especially ragweed) and wool are outstanding inhalant offenders and that other inhalants like atmospheric molds, animals danders, silk, cooking odors, etc., likewise are prom-

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inent offenders. In many of the patients included in that series, a correlation between the specific inhalant to which there was a positive skin reaction and the patient's outbreaks could be obtained. Thus for example, some patients noted increase in itching of the skin after contact with dust. In other instances dust-sensitive patients were relieved by environmental change either to the hospital or seashore. In the latter group, dust desensitization properly carried out was followed by relief. Similar clinical and skin-test evidence emphasized the etiologic relationship of other inhalant allergens to atopic dermatitis.

While all this evidence suggests of course that inhalant allergens are important in atopic dermatitis, it still is purely clinical and open to questionable interpretation. However, the author and his associates[11] have been able to obtain more convincing proof in an experimental clinical study which was recently reported at the meeting of the American Academy of Allergy. In the latter study, atopic dermatitis was induced, apparently for the first time, by inhalation of the offending dust and mold allergens as well as by injection of the specific house dust extract. The study also evoked evidence that the reaction apparently is mediated through the sweating mechanism.

With this demonstration of the causal relationship of inhalant allergens, it becomes evident that in atopic dermatitis as in asthma both inhalant and food allergens can be etiologically significant.

Actually, there should be no surprise that inhalants are important in atopic dermatitis, especially in older children. That there is a close resemblance between atopic dermatitis and allergic bronchial asthma has been emphasized repeatedly by many investigators.[8] Those two conditions have so many characteristics in common, with regard to etiologic factors, diagnosis and treatment, that atopic dermatitis has properly been designated "asthma of the skin." Both conditions frequently coexist or alternate in the same patient. Atopic dermatitis in infancy often is supplanted by asthma of childhood or later life and vice versa. As in asthma, patients with atopic dermatitis are often worse in the winter and especially at the times of seasonal change from summer to fall and from winter to spring. Positive skin reactions are obtained in about the same proportion in both conditions and have about the same clinical significance. Sensitivity to foods is admittedly most important, not only in asthma of infants and young children but also in atopic dermatitis. Likewise, inhalant allergens are more prominent than foods in asthma affecting older children and adults; yet, strangely, the same reasoning has not been applied similarly to its counterpart in the skin.

As in asthma, not all inhalant allergens are equally significant as causal agents. The exact incidence is difficult to ascertain because most of the statistical data depend on skin test evidence. Nevertheless, in addition to the frequency of positive skin reactions, clinical experience also indicated that house dust is a prominent etiologic factor. Thus, for example, some patients will complain of increased itching after dusting; also in asthma, removal of the patient to another environment will be followed by relief. To illustrate:

CASE REPORT

CASE 1: The patient, a girl, aged 16, had flexural atopic dermatitis, recurrent since infancy, and was only symptomatically relieved by the usual local dermatologic management. Allergic study showed positive reactions to dust, wool, pollens, and some foods. The patient was always improved while at the seashore in the summer. This was attributed to the beneficial effect of the sun's rays. She was always worse in early fall and winter months. She was asked to go to the seashore in the spring and upon returning to go not to her own home but to the home of a relative in the same neighborhood. The skin condition cleared up at the seashore and did not recur even in the home of the relative, but it became worse within a day or two after she returned to her own home. Institution of proper dust precautions and desensitization with an autogenous house dust extract in addition to pollen therapy has been followed by pronounced improvement which has persisted for more than three years. It was subsequently determined by clinical trial that none of the foods for which there was positive reaction, with the possible exception of chocolate, were of etiological significance.

Question may arise as to whether, in these patients, the dust excitant acts by inhalation or by direct contact. Since in most patients the lesions involve the flexures, which usually are covered, it is unlikely that the dust will have a chance to act directly, but rather that it operates by inhalation. But if a sufficient amount of dust does come into direct contact with the skin, it can cause difficulty. This was demonstrated clearly in a patient with hay fever and other allergic manifestations who observed that when the house dust present on a dust rag was in direct contact with her skin, it caused pronounced itching. When the dust was inhaled, she first would sneeze or wheeze, and if this reaction was intense, itching of the affected skin would follow. Strict avoidance of contact with dust coupled with injections of the extract has enabled this patient to remain free of dermatitis for more than six years.

Further proof of the importance of inhalation of dust was obtained in the experimental study already mentioned when dermatitis was induced after inhalation of the dust by the patient. It would seem, therefore, that house dust probably is just as important an allergen in atopic dermatitis as in asthma.

Allergy to pollens likewise is an important cause of atopic dermatitis but is apparently neglected in the consideration of the causes of the disease despite reports by Cazort,[1] Feinberg,[3] Rowe[5] and others. There were few of the patients observed by the author who did not have positive reaction to pollen, notably ragweed. In some, the positive pollen reaction can be explained by concomitant hay fever or by a family history of hay fever, and in such instances it has no etiologic relationship to the skin condition. But in many others, it is definitely causal and may induce pronounced aggravation during the season, as for example in the following case:

CASE REPORT

CASE 2: A male, aged 35, was observed in March 1945 with pronounced generalized atopic dermatitis of two years' duration and a history of seasonal hay fever and asthma since the age of nine; also occasional migraine and urticaria occurred and there was a history of hay fever in the father. With the onset of the skin condition in July 1943, there was very little hay fever or asthma—in fact during 1944 only one day of hay fever and asthma. But during August and September 1944, atopic dermatitis was so severe as to require hospitalization for a month with little relief until the hay fever season was over. This experience was so bad that the patient dreaded the approach of another season. Intracutaneous skin tests showed pronounced positive reactions to ragweed pollen and house dust and in lesser degree to other inhalants and foods. Ragweed and dust desensitization was instituted with beneficial results which were still in effect when the patient was last observed in August 1945.

This case report clearly suggests the possible etiologic relationship between sensitivity to pollens and atopic dermatitis. Thus the hay fever and asthma present for many years were replaced almost completely by the dermatitis which was greatly aggravated by the ragweed sensitivity. When the ragweed season was over, the dermatitis was prolonged and became perennial from the same factors, inhalant or otherwise, which operate in perennial asthma secondary to seasonal hay fever.

This tendency for alternation—that is, for one allergic condition to be replaced by another at various times of life—is characteristic of the so-called hay fever-eczema (atopic dermatitis) group of patients, and has been noted by Brocq and many early observers. Sometimes, however, mild outbreaks of the dermatitis appear during the pollinating season, as for example, in the following case report:

CASE REPORT

CASE 3: The patient, a girl five years of age, was first observed at the age of two with atopic dermatitis present since infancy. The parents were told by a pediatrician that the child would grow out of it and only local applications were prescribed. The family history was strongly positive for allergic sensitivity. Positive skin reactions were obtained to house dust, wool, ragweed, milk and a few other foods. The condition was greatly improved by eliminating milk and wool; also by minimizing house dust contact and seashore environment. When first observed the patient had no hay fever symptoms nor was there any flare-up during the ragweed season despite a definite positive skin reaction. But when she was five years of age, while at the seashore in August, she had mild hay fever symptoms which were accompanied by a mild outbreak of atopic dermatitis in the flexures and on the face.

In this case, despite the elimination of the offending allergens from the diet and although the patient was in an environment where she previously had been well, both the dermatitis and hay fever symptoms appeared simultaneously, apparently from inhalation of ragweed pollen. Skin sensitivity to the latter had manifested itself at the age of two, antedating the clinical sensitivity which did not show itself until three years later. This is a good example of a positive skin reaction which represented future or potential clinical sensitivity.

Appreciation of the possible role of pollen sensitivity in atopic dermatitis therefore is essential to the proper management of patients with the disease. Where a possible causal relationship can be established by history or skin test findings or both, pollen desensitization has been definitely beneficial. In the absence of definite history of seasonal aggravation but with positive skin reactions to pollens, it is difficult to decide whether pollen therapy is advisable. If the patient has a positive family history of hay fever or very mild nasal symptoms, the author believes that pollen therapy may be valuable from a prophylactic standpoint even if the pollen sensitivity has no bearing on the skin condition. This opinion is not shared by other allergists.

Sensitivity to atmospheric molds also may be an important etiologic factor in atopic dermatitis, but it is unlikely to be of much significance in the eastern area since the incidence of mold allergy in hay fever or asthma is low there (about 5 per cent) compared with other regions, notably the Middle West. In a series reported by the author, about 15 per cent of the patients with positive reactions to pollens also reacted in slight degree to such molds as alternaria and hormodendrum, but no definite etiologic relationship could be established in that group. However, in the previously mentioned experimental study, atopic dermatitis was induced by the inhalation of alternaria, proving an etiologic relationship.

Sensitivity to animal danders also may be important in atopic dermatitis, but, as in asthma, the exact incidence is difficult to estimate. Thus, many patients have slight or moderately positive skin reactions to feather extract, but it is not always easy to decide whether such reactions were clinically important, first because feather extracts often give non-specific positive reactions and also because clinical relationship could not be easily proved. The patients who have such a reaction probably do not inhale enough feather allergen to produce atopic skin manifestations; nevertheless, sensitivity to feathers may be an additional excitant. Horse, rabbit, cat and dog danders are not prominent causes of atopic dermatitis, probably because of lessened opportunity for contact. Only about 10 per cent of the patients studied had positive skin reactions to extracts of these danders; however, several patients noted definite itching of the affected skin areas or new outbreaks when they were near a cat, dog, rabbit, or horse, even though not in direct contact with the animal. In at least three patients improvement in the skin condition seemed to follow removal of cats to whose dander they had positive reaction. As in asthma, skin tests with extracts of cat, dog, horse or rabbit epithelium are quite specific. When such reactions are present in a patient with atopic dermatitis, diligent search should be made for possible sources of contact.

The importance of wool as an etiologic factor in atopic dermatitis is unquestioned. But whether wool acts as an inhalant or as a contact allergen, or whether it causes aggravation by non-specific irri-

tation, is not always easy to determine. Of the 49 patients studied, 18 stated clearly that itching followed when wool was brought into contact with the skin lesions or even with the normal skin. Undoubtedly, in some of these patients direct contact action is operative and not all of it is due to specific allergic sensitivity. Nevertheless, many patients showed definite improvement when the woolen articles of clothing or bedding were removed completely. Since such articles are not always in direct contact with the lesions of dermatitis, it may be assumed that some of the wool allergen entered the circulation after being inhaled. As with feather extracts, skin tests with wool extract may be non-specific. Thus while 34 of the 49 patients tested had slight to moderate positive reaction, some of the reactions probably were non-specific. A striking instance of wool sensitivity is illustrated in the following case report:

CASE REPORT

CASE 4: A woman 82 years of age was first observed in the hospital with an acute flare-up of generalized dermatitis of the atopic type of several years' duration. Prior to admission the patient had been hospitalized in another institution and was greatly improved until shortly after discharge when, while out on a sun-porch in Atlantic City, she was wrapped in a woolen blanket. This was followed almost immediately by an acute flare-up which again required hospitalization. The patient had eosinophilia (17 per cent) and the family history was positive for atopic dermatitis. Reactions to intracutaneous skin tests were positive for house dust, wool, chocolate and tomato. The patient was greatly improved following the stay in the hospital. She was given instructions regarding avoidance of wool and house dust, but despite this, mild aggravation of the skin condition occurred, due to contact with wool and house dust.

A similar situation exists in relation to silk. Patients with atopic dermatitis may show positive skin reaction to this allergen. Thus 18 of 49 patients tested showed positive skin reaction; in four the reaction was moderate or pronounced. None of these patients apparently noted any evidence of clinical sensitivity, although this is not easy to detect. However, as previously mentioned, evidence that silk can be responsible for aggravation of symptoms in patients with atopic dermatitis has been furnished previously; while it had been believed that, as with wool, the reaction is provoked by direct contact of the silk with the patient's skin, the experiments of Sulzberger and Vaughan[9] conclusively demonstrated that silk protein when inhaled is absorbed into the circulation and thus can aggravate or induce the lesions of atopic dermatitis. It is not always possible to determine in a silk-sensitive patient which route is culpable. Reactions to patch tests frequently are negative, but this does not exclude possible contactant action. Regardless of the method of action, it is advisable for patients with positive reaction to silk to remove all possible sources of silk contact.

Other inhalant allergens like orris root, tobacco, insecticides, goat hair, cereal flours, etc., are possible but infrequent causes of atopic dermatitis. Thus for example, occasional instances have been reported of atopic dermatitis secondary to inhalation of cereal flours, especially in bakers, but these are the exception rather than the rule. Even the inhalation of the odors of cooking, especially of fish, or of perfume may cause aggravation in some patients. Interest again has been revived in the possibility that sensitivity to human dander is a causative factor in atopic dermatitis, especially in infancy. Allergic reaction to human dander was first described in 1925 by Van Leeweun[12] who considered it important in bronchial asthma. But this was never clearly proved and the subject was dropped. Recently Simon[7] reported positive skin reactions to human dander and positive results of passive transfer tests in infants with atopic dermatitis. He postulated that in such instances the human dander present in the scalp of the mother or father may aggravate the lesion either by direct contact with the skin or as a result of inhalation. No definite etiologic relationship has been established as yet between the dermatitis and sensitivity to human dander.

As previously mentioned, allergic sensitivity to food long has been considered the major etiologic agent in atopic dermatitis, probably because the sensitization is hematogenous and food allergens are known to be absorbed into the blood after ingestion. As early as 1915, Schloss[6] attributed infantile eczema to such foods as eggs, milk and cereals. His observation subsequently was corroborated by many others and especially by Hill[4] who showed that in young children with atopic dermatitis, removal of allergenic foods, particularly eggs, from the diet was followed by pronounced improvement or even cure, and that reintroduction of them into the diet brought about a recurrence. There is little question, then, that food sensitivity is important in infants and young children. It is only in older children and adults that its significance has been doubted, and, as already has been emphasized, this is explained by a failure to consider the inhalant allergens in the older age groups. This does not mean that food sensitivity is of no importance in older children or adults. It bears the same relationship as in asthma, where elimination of foods to which there is specific reaction is followed by relief and the reintroduction of them by aggravation. The author has found this as true with regard to adult patients with atopic dermatitis as it is to those with asthma.

The specific food allergens most frequently responsible for atopic dermatitis are well known. Egg is especially prominent. Sensitivity to egg should be suspect in all cases of atopic dermatitis even when the reaction to a skin test is negative.

CASE REPORT

CASE 5: A man 40 years of age had atopic dermatitis that had begun as a generalized eruption in infancy, then had become flexural in distribution with repeated recurrences since. The eruptions were more persistent on hands and face. Early and late hay fever complicated by mild asthma had occurred since the age of 12, but these symptoms had been greatly lessened by specific pollen therapy. Over the years, considerable and varied treatment had been given for the dermatitis, including local medicaments, x-ray, sunlight,

fever therapy, and more recently psychotherapy with only temporary beneficial effect. The patient had been tested previously but was told that foods were of no etiologic importance.

When first observed by the author, the patient had definite areas of atopic dermatitis on the cheeks, about the mouth, on the flexures and especially on the wrists and hands.

Intracutaneous skin tests revealed significant positive reactions to house dust, feathers, wool, tobacco, timothy, ragweed, English plantain, cornmeal, wheat, milk, cheese, chocolate, tomato, and a few other vegetables. On repeated testing, reaction to egg was negative.

Elimination and avoidance of positive inhalants was followed by only slight improvement. The patient then was put on a trial diet and after ten days to two weeks showed striking improvement. However, when egg was added to the diet, a pronounced flare-up occurred. When the dermatitis had subsided, another trial with egg was followed by a flare-up. Since then, no egg has been taken and the skin condition has been quite good except when the patient eats proscribed foods.

This case report illustrates very well the fact that patients with atopic dermatitis, even adults, may have allergic sensitivity to certain foods even though reaction to skin tests with allergens of them are negative. It emphasizes the importance of diet trial in such patients, especially if elimination of test-positive allergens does not relieve the condition.

Probably next in frequency to egg among the causative foods are milk and wheat. While any food allergen may be important, the exact incidence or the relative etiologic significance of any particular food is difficult to estimate because this information depends upon skin test reaction rather than upon clinical evidence or diet trial.

In addition to inhalant and food allergens, atopic dermatitis, like asthma, may be caused or aggravated by other factors, including bacterial, physical and psychosomatic.

The etiologic significance of bacterial agents is hard to estimate because it is difficult to establish an etiologic relationship. Bacterial sensitization from an infected focus (e.g., tonsils or teeth) may be responsible for atopic dermatitis. But this is even harder to prove in patients with atopic dermatitis than it is in those with asthma, since skin tests for sensitivity to bacteria are unreliable and even constitutional reaction from injected vaccine is less likely to provoke atopic dermatitis than it is to cause asthma. Proof is made additionally difficult by the fact that atopic dermatitis generally is worse in the winter or at times of seasonal change (summer to fall or winter to spring) when upper respiratory infections are prevalent. But (as was explained in the discussion of asthma) symptoms similar to those of infection in the upper respiratory tract may be due to allergic rhinitis provoked either by atmospheric changes or by excessive exposure to environmental allergens (e.g., dust or wool). Bacterial sensitization therefore is a possible secondary or aggravating factor but seldom is primary in invoking atopic dermatitis.

Atopic dermatitis also may be aggravated by various physical factors such as heat, cold, and physical exertion, just as is the case with asthma. In fact, any influence tending to increase skin irritability or to stimulate skin congestion and sweating also will either increase the tendency toward or actually aggravate already existing dermatitis. This applies to bacterial toxins or toxic products absorbed from the gastrointestinal tract, as in so-called "auto-intoxication"; also, probably, to such factors as fatigue, overfeeding or even dentition, mentioned often as a possible cause of atopic dermatitis. Experimental studies of this condition[11] indicated it is very likely that disturbances of the sweating mechanism may initiate or aggravate dermatitis.

The significance of psychosomatic influences in the production of atopic dermatitis likewise is a moot question. That they might be important is indicated by the continued use, especially by dermatologists, of the term "neurodermatitis" although it originally was intended to indicate vasomotor instability and not neurogenesis or psychogenesis. Unquestionably, emotional upsets and perhaps hidden or subconscious influences of various sorts can cause aggravation of the skin lesions. But, as in asthma, it is doubtful that such factors can be responsible primarily or entirely for the condition. The outstanding primary symptom of atopic dermatitis is itching, which causes the patient to scratch. This leads to subsequent thickening of the skin and explains the secondary excoriations and other changes in the skin. Undoubtedly, the degree of itching and secondary change will be greater in the overanxious, emotionally unstable psychoneurotic patient, but this does not prove necessarily that the primary factor responsible initially for the itching is psychosomatic. Another item of evidence against primary psychogenesis is the fact that this condition so often starts in infancy or early childhood, an age period in which psychosomatic influences supposedly seldom operate.

### CLINICAL MANAGEMENT

As with other allergic diseases, treatment of atopic dermatitis may be specific, non-specific and/or symptomatic. The specific treatment of atopic dermatitis will depend largely upon whether the causative factor has been ascertained. Complete allergy study, including history, tests for sensitization and diet trial, is essential. Despite the current confusing reports, all patients should be tested for allergic reaction either by the scratch or intracutaneous method or, if this is not possible, as in eczematous infants, by the indirect or passive transfer method of Walzer. Patch tests are valueless from a practical standpoint and although study of them has been of some academic importance, they should not be used in the determination of offending excitants.

If the specific allergens to which the patient is sensitive can be determined, avoidance of contact with them or elimination from the diet, as the case may be, often will be followed by improvement or complete relief, especially if a specific food such as egg, milk or wheat is the principal excitant. Complete elimination of the latter foods is essential if the patient has allergic sensitivity to them. Due care

should be taken to see that no foods are taken which contain these elements even in small amounts. Removal of milk from the diet of infants may seem difficult, but efficient substitutes are available.

The striking benefits which sometimes follow food elimination are exemplified in the following case report:

CASE REPORT

CASE 6: A male child, 22 months of age, was first observed in June 1949 with "eczema" which had been present since early infancy. Some improvement had followed milk elimination but in April 1949 there was pronounced increase with subsequent progression. Mild attacks of rhinorrhea and slight wheezing were noted in June 1948. Family history was strongly positive for allergic disease; the mother was being treated for hay fever. When first observed, the child had severe atopic dermatitis affecting especially the flexures.

Reactions to intracutaneous skin tests were positive to house dust, ragweed pollen and also to milk, wheat, chocolate, green pea, tomato and string bean.

Following the initial examination and because of the history, milk was eliminated completely. This was followed by slight improvement. Later, when the tests were finished and all the test-positive foods eliminated, the dermatitis cleared completely. The mother noted that this was especially striking after tomato was taken away. This improvement has been maintained except for a mild outbreak after the ingestion of string bean given for purposes of diet trial and also after the ingestion of chocolate by the child unknown to the mother.

It is to be notetd that in this patient recovery followed elimination of foods alone and that, so far, desensitization procedures have not been necessary. But if there is recurrence which can be traced to inhalants, injections of the appropriate extracts will be given.

When specific food excitants are not revealed by the usual diagnostic methods or even during the course of further study, the patient should be placed on an elimination or trial diet for a period of one to two weeks, then individual foods added and the effect upon the eruption noted. If improvement follows, the diet trial should be continued until the eruption disappears. If not, other diets should be tried in order to determine definitely whether sensitivity to food is a factor. It must be remembered that the improvement in atopic dermatitis is much slower than in asthma; hence, the method of treatment should not be changed until sufficient time has been allowed for improvement to take place following the withdrawal of a certain food. Dietary restriction should not be too rigid in infants or young children with numerous positive reactions; it is preferable to exclude first the most important of the items to which there was positive reaction. Sufficient minerals and vitamins also should be added to compensate for any diminution caused by the dietary restriction.

Elimination of offending inhalants is just as important for patients with atopic dermatitis as it is for those with asthma. This may be easy with some allergens, such as feathers, and difficult with others such as house dust. If wool is a factor, the patient should be told either to provide substitutes (e.g., a cotton quilt for a woolen blanket) or to keep the wool covered in such a way as to prevent its coming into direct contact with the skin (as by lining woolen snowsuits for children with cotton sheeting) or from being inhaled (as by encasing a woolen blanket in a heavy cotton blanket cover). When inhalant contact cannot be completely avoided, specific desensitization may be required. This is true especially with regard to house dust, pollens and molds. Desensitization with extracts of animal danders or wool is not very effective but seldom is necessary.

Care should be exercised in the desensitization procedures, especially with house dust and pollen extracts. Constitutional reactions are slower in dermatitis than in asthma or hay fever and are indicated by aggravation of the dermatitis in 24 hours or longer rather than by acute flare-up. For example, a patient was being given injections of an autogenous house dust extract, and when the point of maximum tolerance was reached, further injections were followed in a day or two by new outbreaks of dermatitis. The injections were stopped and the skin cleared up. Later, the injections were begun again in lower dosage which was tolerated much better.

It is extremely important for the attending physician to recognize the delayed nature of reactions to these injections which might be responsible not only for aggravation of the dermatitis but also for persistence of it.

Non-specific measures sometimes may be required if specific treatment fails or if progress is slow. In the author's experience, however, nearly all our patients respond to specific treatment. Among the most often used of non-specific measures is autohemotherapy, which occasionally may be helpful in stubborn cases. Vitamin injections (notably vitamin B) are used extensively by dermatologists who believe them to be helpful in chronic cases. The author has had no personal experience with them nor observed indication for their use. Bacterial vaccines, either stock or autogenous, have a very limited application although some physicians claim good results with autogenous vaccines prepared from various foci of infection. Injections of histamine in increasing dosage are commonly used, but as in asthma the beneficial effect is limited and difficult to judge. But it is worthy of trial in selected cases. The author sometimes finds it useful as a substitute for other injections given for purposes of desensitization (e.g., house dust extract) especially when the latter is suspected of inducing reaction or producing aggravation of the existing dermatitis.

Regardless of whether specific or non-specific treatment is required, symptomatic or palliative measures usually are necessary even during the period of diagnostic study, to relieve the itching. These include local remedies to aid in the disappearance of the lesion. It often is surprising, however, how little local treatment may be required for patients in whom the causal factor has been found

and eliminated. Just as it is the wheezing which requires relief in asthma, in atopic dermatitis it is the itching. For itching causes repeated scratching which in turn aggravates the lesion and is responsible for excoriation and secondary infection and thickening of the skin. To reduce the damage of scratching, the nails should be kept short and the fingers clean to prevent secondary infection. If possible the lesions should be kept bandaged day and night to prevent access to them. Cardboard cuffs over the elbows and forearms may be necessary. The arms and legs may have to be tied to the side of the crib or bed.

The itching may be reduced by various local applications of soothing agents. For this purpose, it is well to obtain the advice of a dermatologist. Antipruritic lotions are helpful, especially if there is much itching and oozing. Bland ointments like Lassar's paste or those containing small amounts of crude coal tar likewise are beneficial. Soaps should be avoided and the skin cleansed either with olive oil or mineral oil or with soapless detergents. Roentgen-ray or ultraviolet therapy may be helpful in some cases but decision with regard to it is best left to a dermatologist.

Sedatives—bromides, barbiturates, chloral—may be used to relieve intense itching or to calm highly emotional patients. They may be given at regular intervals during the day, but especially at bedtime when the excessive warmth of bedclothes often increases itching. Coincident psychotherapy naturally should be given those patients requiring it, but in many instances calm reassurance will be sufficient.

The necessity for the use of psychotherapy seems to be inversely proportionate to the ability of the attending physician to find the cause of the dermatitis and to treat it successfully by specific measures. If specific cause is not found the patient is considered to be psychoneurotic and is treated accordingly. Then it may be noted that the dermatitis improves strikingly when the patient takes a vacation trip away from home in order to rest his "tired nerves," only to recur when he gets back. Recrudescence then is attributed, naturally, to the return to an environment conducive to emotional upset. That leaving home may also mean leaving environmental allergens is too often overlooked in such instances.

The antihistaminic drugs have been used extensively in the treatment of atopic dermatitis, but, as in asthma, it is difficult to evaluate correctly the statistical reports. Some patients obtain relief of the itching from these drugs given in full dosage orally three or four times daily. With so many drugs available, selection of one that is more effective than another would be difficult. The author has given Benadryl® or Pyribenzamine® with varying results; some patients benefited temporarily and others noted no benefit. It is the author's impression that the antihistaminics were more effective in patients with associated nasal symptoms than in those without them, which is to be expected if inhalants are responsible for the dermatitis. In the experimental study previously mentioned, it was found that Benadryl administered either by intramuscular or intravenous injection in doses from 20 to 50 mg. produced better results than it did when given orally. If the dermatitis was mild, it subsided more quickly with the drug than without; if severe, subsidence was slower. This is comparable perhaps to the effect of epinephrine in asthma. At any rate, it appears that antihistaminics may give symptomatic relief in some patients with atopic dermatitis and are worthy of further trial.

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